EMERGENCY CASEBOOK

A case of miliary tuberculosis presenting with bowel perforation

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Tuberculosis is a disease that should never be underestimated. It can affect anybody at any age. Doctors in the West do not have much experience of peritonitis secondary to tuberculosis. It is a condition that requires urgent and aggressive management as it can be fatal, even in the young and fit, as this case report illustrates.

A 25-year-old woman of Pakistani origin presented to the Accident and Emergency Department with a 1-day history of progressively worsening central abdominal pain. This was preceded by 4 months of non-productive cough, night sweats, poor appetite and weight loss. She had not been vomiting. There were no past medical problems and she was not taking any drugs. She denied exposure to tuberculosis and had received BCG vaccination as a child. She was born in Pakistan and had lived there until 4 years previously.

On examination, she appeared ill and cachectic with scattered crepitations present throughout both lungs. Her abdomen was rigid, with generalised tenderness. Bowel sounds were absent. Her heart rate was 130 beats/min and blood pressure was 98/65. Haemoglobin was 9.1 g/dl and white cell count was 3.8×10^6 . An erect chest radiograph showed patchy consolidation of both lungs and a large amount of air under the diaphragm.

An emergency laparotomy was carried out. One litre of blue-green fluid was discovered on opening the peritoneum. The caecum and terminal ileum were inflamed, with adhesions between the two structures. The site of perforation was identified at the terminal ileum, proximal to a stricture 10 cm from the ileocaecal valve. The mesenteric lymph nodes were enlarged. The terminal ileum (20 cm) was resected, and a right hemicolectomy was carried out, followed by side-to-side stapled anastomosis. A few mesenteric lymph nodes were excised, with bisection showing caseous necrosis.

Postoperatively, this patient was transferred to the intensive treatment unit and treatment for tuberculosis was started. Unfortunately, she died 6 days later from acute respiratory distress syndrome and disseminated intravascular coagulation.

HISTOLOGY

Transmural inflammation of the ileum and caecum with mucosal oedema and ulcerated patches was found, producing thickening of the caecum. Fat wrapping had taken place at the caecum. Serosal pustules measuring up to 4 mm were seen. The perforation was 2×2 mm in size.

On microscopical examination, mucosal ulceration with transmural acute and chronic inflammatory cell infiltrate was seen, together with well-circumscribed areas of necrosis. The muscularis propria was destroyed in places. Ziehl-Neelsen staining confirmed the presence of acid-fast bacilli. The

mesenteric lymph nodes showed lymphoid tissue largely replaced by necrosis.

DISCUSSION

Mycobacterium tuberculosis is responsible for almost all cases of abdominal tuberculosis. Infection with *M bovis*, found in dairy products, is reported in some parts of the world but is uncommon in Western countries.¹ There has been a resurgence in the West owing to HIV.² Primary abdominal tuberculosis due to ingestion of contaminated food is very rare.² Secondary intestinal disease arises from swallowed sputum leading to the direct penetration of the mucosa by the organism, or during a bacteraemic phase in patients with pulmonary tuberculosis,² in which case the peritoneum, mesenteric nodes and the intestines may become infected along with other abdominal and pelvic organs.

The most common site of intestinal tuberculosis is the terminal ileum and the ileocaecal junction. Other sites in order of incidence are the ascending colon, jejunum, appendix, duodenum, stomach and oesophagus.¹⁻⁴ Patients may also present with abdominal pain, diarrhoea and blood in the stools along with symptoms of pulmonary tuberculosis. The most common symptoms are anorexia, abdominal pain and weight loss.³ A mass may be palpable in rifabutin. Patients are usually anaemic, with a normal white cell count. Complications include obstruction, perforation, haemorrhage and fistula formation.^{1,2}

Diagnosis is aided by keeping such possibilities in mind, given the appropriate risk factors and symptoms. A definitive diagnosis is made by identification of acid fast bacilli in the tissue or by culture. HEAF test cannot confirm or exclude active disease.² A chest radiograph may show apical or miliary lesions. Computed tomography may show a thickened ileocaecal valve and lymphadenopathy,² and is better at identifying abnormalities than an ultrasound scan.⁴

Figure 1 Site of perforation at the terminal ileum.

A macroscopic examination can show ulcerative lesions, hyperplastic lesions and sclerotic lesions.^{1 2} Multiple tubercles may be visible on the serosal surface. Ulcers, which are commonly seen, are usually transversely placed.² In the case of strictures, obstruction or perforation, treatment is by surgical resection. Otherwise, a 12-month treatment with a tuberculous regimen is usually satisfactory. Unfortunately in this case, the patient's late presentation and advanced stage of disease meant that she lacked the physiological reserve to cope with this pathological insult.

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